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Guidelines for Surface Water Quality

Vol. 1 Inorganic Chemical Substances

ARSENIC

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Abstract

A literature survey was carried out on the toxic effects of arsenic and arsenic compounds on human health, aquatic life, plants and livestock. The information is summarized in this publication. From it, maximum arsenic concentrations in water at which toxic effects will not appear are recommended.

Résumé

On a effectué une étude de la documentation portant sur les effets toxiques de l'arsenic et de composés de l'arsenic sur les humains, les organismes aquatiques, les plantes et le bétail. Ces données sont résumées dans cette publication. En se basant sur ces données, on propose des valeurs des concentrations maximales d'arsenic dans l'eau auxquelles on n'observe pas d'effets toxiques.

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RECOMMENDED WATER QUALITY OBJECTIVES FOR ARSENIC

Uses	Recommended objectives
Raw public water supply	0.05 mg/L as total arsenic
Aquatic life and wildlife	
Aquatic life	Indications from the literature suggest that 0.05 mg/L, the suggested limit for raw public water supplies, should protect organisms frequenting natural fresh waters
Wildlife	No information available
Agricultural water supply	
Livestock watering	0.5 mg/L as total arsenic
Irrigation	Sensitive crop: sandy loam, 0.2 mg/L as total arsenic; clay, 1.0 mg/L as total arsenic Tolerant crop: sandy loam, 1.0 mg/L as total arsenic; clay, 2.0 mg/L as total arsenic
Recreation and aesthetics	
Recreation	0.05 mg/L as total arsenic
Aesthetics	No recommended limit
Industrial water supplies	
Food processing	0.05 mg/L as total arsenic

Guidelines for Surface Water Quality
Vol. 1. Inorganic Chemical Substances. Arsenic

Adrian Demayo, Margaret C. Taylor and Stewart W. Reeder

OCCURRENCE, FORMS AND SOURCES

Natural Occurrence

Arsenic ranks as the 53rd element in abundance in the earth's crust with an average concentration of 1.8 mg/kg (Taylor 1964). Arsenic is more common in the earth's crust than other well-known elements such as mercury, iodine, cadmium and silver. In sea water arsenic concentrations range between 0.00015 and 0.006 mg/L, with an average value of approximately 0.002 to 0.003 mg/L (Woolson 1975; Bowen 1966, pp. 19-20). Arsenic minerals are widely distributed, the most common being those in which the arsenic is combined with sulphur and iron or nickel, e.g., realgar (As_2S_2), mispickel (FeAsS), nickel glance (NiAsS) (Smith 1973, p. 547; Luh *et al.* 1973).

The arsenic content of natural fresh waters varies over a wide range depending on the geochemistry of the region, the proximity of industrial works and other human activities. In Canada, the dissolved arsenic concentration in over 5400 river water samples collected between 1972 and 1977 ranged from less than 0.001 mg/L to approximately 0.050 mg/L, with 90% of the results being below 0.008 mg/L (WQB 1977). Lake water samples collected during the same period had arsenic concentrations ranging from less than 0.0005 mg/L to approximately 0.020 mg/L (approximately 900 results) (WQB 1977). In the Great Lakes, the arsenic concentration ranged from $<$ 0.0001 to 0.0012 mg/L in water, from 0.5 to 14.0 mg/kg in sediments, and from 0.03 to 0.12 mg/kg in fish. The rivers in the Great Lakes region had arsenic concentrations of $<$ 0.0001 to 0.0014 mg/L (Traversy *et al.* 1975). Some high arsenic concentrations reported in surface waters around the Yellowknife area (Northwest Territories) were 0.135 mg/L in Fault Lake, 2 to 3 mg/L in Kam Lake, and 12.6 mg/L in Baker Creek (CPHA 1977, p. 53; WQB 1977). In some thermal waters in Wyoming, Nevada, California, etc., the arsenic concentrations ranged from 0.020 to 3.8 mg/L, reaching a high of 198 to 243 mg/L in Searles Lake, California (Woolson 1975).

The arsenic concentration in the sediment in the Puget Sound estuary (State of Washington, U.S.A.) was strongly correlated with antimony and carbon concentrations, and fine particle size (Crecelius *et al.* 1975). The same study suggested that all arsenic in the sediments came from the river particulates. The river particulates (>0.45-μm size) carry one third to two thirds of the total arsenic load of the river (Crecelius *et al.* 1975). Some high arsenic concentrations, up to 2400 mg/kg, were reported in sediments from around the Yellowknife area (e.g., Back Bay around Baker Creek inflow) (CPHA 1977, p. 51).

Munro (1976) reported that freshwater fish (pickerel, pike, smelt, whitefish) caught in Canada contained arsenic concentrations of 0.2 to 0.5 mg/kg. Concentrations of the same order of magnitude, 0.3 to 0.8 mg/kg, were found in Atlantic salmon, bluefin tuna, and herring. Concentrations in marine bottom feeding fish (cod, halibut, flounder) were higher, ranging from 2.5 to 4.9 mg/kg with grey sole having a concentration of 56.4 mg/kg. Arsenic concentrations were 1.2 mg/kg in clams and scallops caught in Canadian marine waters, 5.9 mg/kg in lobster, and 10.9 mg/kg in shrimp.

Forms of Arsenic

Arsenic can exist in the valence states of −3, 0, +3 and +5. It forms a wide range of compounds with a large number of elements. The two most common valence states are +3 [As (III)] and +5 [As (V)]. Arsenic (V), as arsenate (AsO_4^{3-}), is the stable form in aerobic waters. Arsenic (III), as arsenite (AsO_3^{3-}), may be present in surface waters if the oxidation to As (V) is incomplete. Arsenic (III) is the predominant form under anaerobic conditions (Carriker *et al.* 1976). It has been suggested that bacterial reduction can convert arsenate to arsenite in the open ocean and surface waters (Johnson 1972). Calculations have shown that in the presence of barium or possibly chromium ions, the dissolved arsenic concentration in fresh waters is likely to be very small (Wagemann 1978). Thus, at pH 8 and a barium free ion concentration of 0.03 mg/L the calculated dissolved arsenic concentration in a "typical" fresh water was 2.4×10^{-7} mg/L. Similarly, at pH 8 and a chromium free ion concentration of 0.001 mg/L the calculated dissolved arsenic concentration was 0.00011 mg/L. Arsenic (III) has a strong affinity for sulphur and it readily adsorbs and coprecipitates with other metal sul-

phides. Arsenic (V) as arsenate adsorbs on hydrous iron oxides, aluminum hydroxide, and clays. Both As (III) and As (V) form stable bonds with carbon, resulting in thousands of organo-arsenical compounds, some of which are very toxic (Ferguson and Gavis 1972). An important group of such compounds, from an environmental point of view, is the methylarsines, which can be formed by biological methylation of inorganic arsenic compounds (Ferguson and Gavis 1972; Brinckman and Iverson 1975). Note that in arsines the oxidation state of arsenic is -3.

Dimethylarsinic acid, $(CH_3)_2AsOOH$, is a major and ubiquitous form of arsenic in the environment. It is very resistant to oxidation, and as such, it could have a long residence time in natural waters. The toxicity of dimethylarsinic acid is approximately 25 times less than that of As (III) (Braman and Foreback 1973). Dimethylarsinic acid and methylarsonic acid, $CH_3AsO(OH)_2$, were found in a wide range of natural waters, e.g., rivers, lakes, ponds and sea in and around Tampa, Florida (Braman and Foreback 1973). Biomethylation to trimethylarsine and dimethylarsine as the main products has been observed in soils treated with various arsenic compounds (Braman 1975b). Arsenic (III) also forms stable bonds with sulphur in organic compounds. It reacts with the sulphydryl groups of cysteine in proteins, leading to enzyme inactivation. This is the main mechanism for arsenic toxicity. A summary of environmental chemistry of arsenic has been presented by Braman (1975a). Wood (1975) has also proposed a scheme for a "natural" arsenic cycle involving the air, water and sediment in which the arsenic is reduced, oxidized, methylated, etc., depending on the environmental conditions (e.g., aerobic, anaerobic, pH) and the bacteria present. This cycle has been reproduced in Figure 1.

Uses of Arsenic

In early Chinese and Greek civilizations arsenic compounds were known and used as pigments, therapeutic agents, and poisons. Arsenic compounds have been used for many years in treating various diseases, syphilis in particular. At present antibiotics have replaced most of the arsenical drugs.

Arsenic is used in glassmaking (decolourizing and firing of glass products) and in alloys with lead and copper. For example, the addition of small amounts of arsenic, up to 3%, hardens lead; copper with 0.15% to 0.50% arsenic is recommended for high-temperature applications. Arsenic compounds (e.g., calcium and lead arsenates, sodium arsenite) are used as pesticides, weed killers, cotton and potato plant defoliants, and wood preservatives. Arsenic compounds are also used in hide tanning and in the manufacture of some paint pigments. High-purity arsenic, >99.999%, is used for the manufacture of semiconductors (George 1970; Smith 1973).

In Canada, the available data for the year 1974 (Statistics Canada 1976a,b,c) show arsenic and arsenic compounds being used in glass manufacture (162 tonnes); in metal rolling, casting and extruding (76 tonnes); and in miscellaneous chemical industries (2 tonnes).

Pathways for Entering the Environment

It has been estimated that the arsenic entering the environment by weathering amounts to approximately 45 000 tonnes per year. No numerical value could be found in the literature for the amount of arsenic released to the environment by volcanic activity, another natural phenomenon, but it has been estimated to be smaller than the contribution through weathering (Ferguson and Gavis 1972).

The contribution of arsenic to the environment by human activities is probably much higher than by natural phenomena. The world production of arsenic is around 50 000 tonnes per year, of which a large part reaches rivers, lakes and oceans (Ferguson and Gavis 1972). In addition, arsenic is released to the atmosphere during the processing (smelting, roasting) of any mineral containing sulphide and in the combustion of fossil fuels, especially coal. The presence of arsenic in most pyrites leads to arsenical contamination of commercial sulphuric acid produced by roasting pyrites in the "chamber process." Additional contamination is caused by the sulphuric acid used in the manufacture of other chemicals. For example, certain laundry products have been shown to contain up to 36 mg/kg arsenic (Luh *et al.* 1973). Ferguson and Gavis (1972) have estimated the annual amount of arsenic released to the environment as a result of human activities to be around 110 000 tonnes or approximately three times the amount reaching the environment from weathering. Most of the arsenic reaching the environment is sorbed by soils and sediment (Carriker *et al.* 1976).

In Canada the quantity of arsenic produced is relatively small. The production of arsenic in 1970 was approximately 49 tonnes, and in 1971, 34 tonnes. After 1971 the production decreased even further to almost zero (Statistics Canada 1976d). Stack emissions of arsenic in Canada in 1972 have been estimated at approximately 3695 tonnes. The largest contributor was metallurgical processing in the gold industry (47.5%), followed by primary iron and steel production (25.6%), primary copper and nickel production (16.2%), and primary zinc production (8.8%). The contribution from other sources was very small: fuel combustion, about 1% and solid waste incineration and pesticide appli-

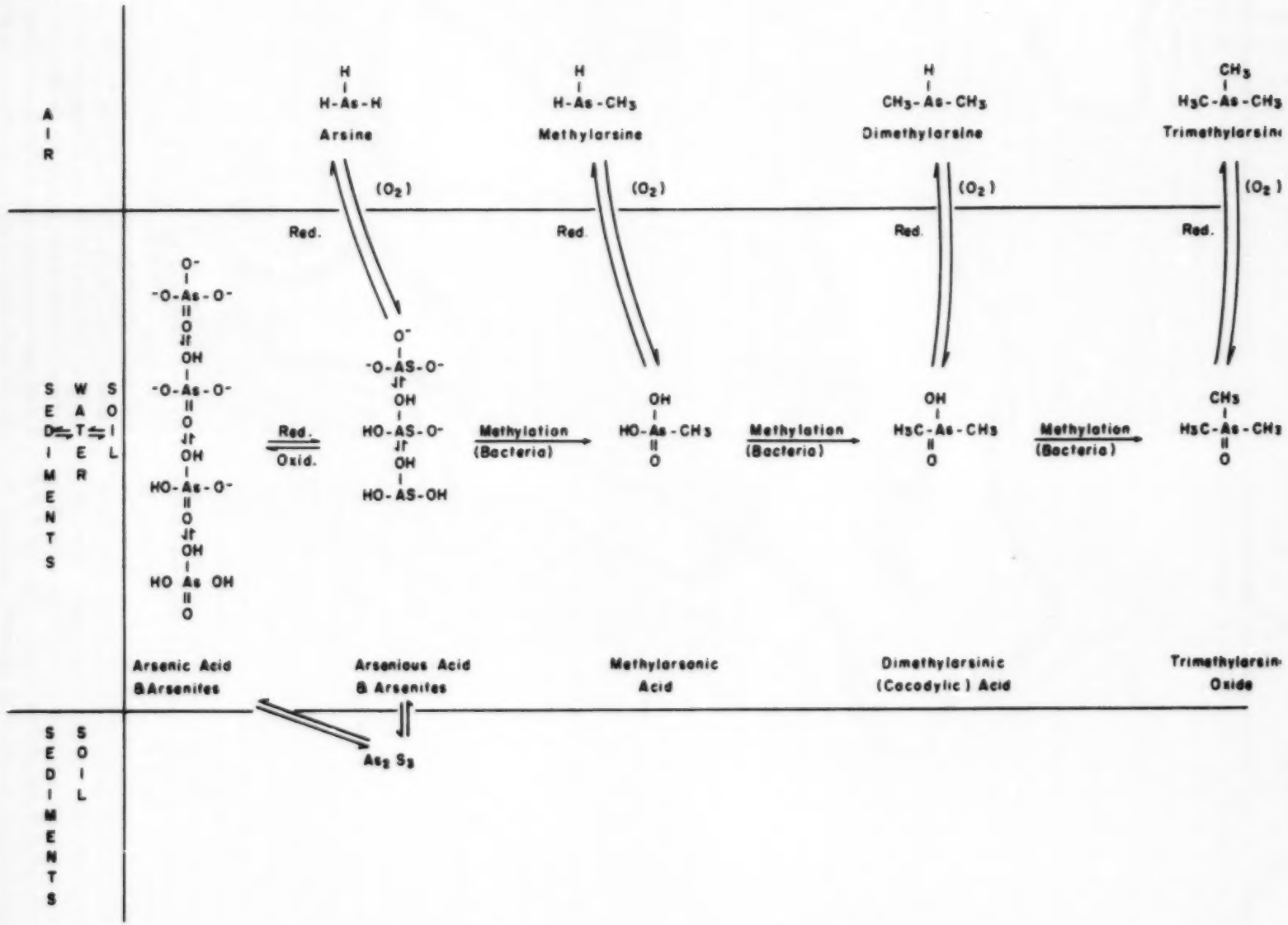

Figure 1. The arsenic cycle (adapted from Wood 1974, 1975; Braman 1975a; McBride and Wolfe 1971).

cation, around 0.2%. Geographically the emissions were: Ontario (71.7%), Manitoba (12.5%), and Quebec (10.2%) (Environment Canada 1976).

RATIONALE

Raw Public Water Supplies

Toxicity of Arsenic to Humans

Acute intoxication, generally resulting from the ingestion of an arsenic compound, produces severe hemorrhage of the stomach or the small intestine, leading to gastrointestinal bleeding, dehydration, electrolyte imbalance followed by renal shutdown, cardiovascular collapse, and death. Acute intoxication also results in a high concentration of arsenic in the central nervous system (Wagner 1972). Ingestion of 100 mg of arsenic has resulted in severe poisoning and 130 mg has proved fatal to humans (Browning 1961).

Arsenic compounds generally enter the human body via the gastrointestinal and respiratory systems. The symptoms connected with chronic arsenic poisoning are nonspecific weakness, malaise, and vague abdominal pains. Long-term environmental exposure and occupational exposure lead to hyperpigmentation and/or dermatitis, chronic ulceration of the skin or nasal septum, and peripheral neuritis (Wagner 1972; Smith 1972). Arsenic compounds have also been implicated in skin, respiratory system, and liver cancers as well as in "Blackfoot" disease, a gangrene of the feet secondary to severe peripheral arteriosclerosis (Hoffmann and Wynder 1976; Groth *et al.* 1976; Jackson and Grainge 1975; Wagner 1972). The induction period from the beginning of ingestion to the onset of carcinogenic lesions owing to chronic arsenic poisoning is very long, 24 to 50 years (Wagner 1972; Jackson and Grainge 1975). Reports of chronic arsenic poisoning in persons ingesting 3 to 6 mg As/day were quoted by Lisella *et al.* (1972). Von Sagner (1973) reported chronic arsenic poisoning in a patient ingesting 2 mg As/day for three years. Several incidents of chronic poisoning by drinking water with arsenic concentrations ranging from 0.21 to12.2 mg/L were quoted in the literature (Lisella *et al.* 1972; Aston *et al.* 1975; Jackson and Grainge 1975; Penrose 1974). Goldsmith *et al.* (1972) concluded that although no medical condition was reported, drinking water containing more than 0.05 (±0.03) mg As/L led to accumulation of arsenic in the body, which was reflected by increased levels of arsenic in hair. Although apparently in humans, arsenic does not accumulate in organs such as liver, kidney or skin, exposure to arsenic has a cumulative effect on some body tissues. Elevated excretion of arsenic and elevated levels in hair and nails are some indications of chronic arsenical intoxication (Wagner 1972).

The degree of toxicity of arsenic compounds depends on their chemical form. Using several references, Penrose (1974) gave an approximate order of toxicities of arsenic compounds. The most toxic were the inorganic and organic arsines, followed by arsenite, arsenoxides, arsenate, arsonic acids and other As(V) compounds, arsonium compounds, and finally, metallic arsenic. In the body As(III) reacts with –SH groups of various proteins and enzymes such as glutamic oxaloacetic transaminase, pyruvate oxidase, monamine oxidase, choline oxidase, glucose oxidase, and urease (CPHA 1977, p. 75).

In addition to the chemical and physical (e.g., solubility) properties of various arsenic compounds, the toxicity of arsenic depends also on exposure route, dose and duration of exposure.

Humans who consumed shrimp containing 120 mg/kg arsenic excreted all the arsenic in 4 days. Similar results were obtained with rats. With rats fed As_2O_3, only 21% of the arsenic was excreted and the liver contained 20 times more arsenic when compared with the liver of rats on a shrimp diet (Coulson *et al.* 1935).

Experiments with adult swine, in whom the physiology of the digestive system is similar to that of humans, have shown that 90% of the arsenic was eliminated in 3 days following a single meal of fish containing arsenic. In investigations with adolescent monkeys (*Maccaca iris*), only 63% of the administered arsenic was recovered, suggesting that the monkeys had retained part of the ingested arsenic (Munro 1976). These results point out that the retention of arsenic depends on the organism involved and the chemical form of the arsenic.

Recommended Objective for Raw Water Supply

Data quoted by the Canadian Public Health Association (CPHA 1977, p. 70) estimate the human arsenic intake in the normal diet at between 0.020 and 2.0 mg/day. According to the World Health Organization, the maximum acceptable load of arsenic is 0.05 mg/day·kg of body weight. Therefore, for a 70-kg person, the maximum acceptable arsenic intake would be 3.5 mg/day, which seems to be very high (CPHA 1977, p. 51). The estimate for the average intake of arsenic from Canadian diets quoted in the same reference is less than 0.100 mg/day. Smith *et al.* (1973) estimated that the dietary arsenic intake was less than 0.030 mg per person. This estimate agrees with the estimated daily dietary intake of arsenic in the United States of 0.020 mg (Jelinek and Corneliussen 1977). The proposed *Drinking Water Criteria for Canada* recommend a maximum permissible limit of arsenic in drinking water of 0.05 mg/L; based on the fact that there was no evidence of any specific illness associated with ingestion of drinking

water containing 0.05 mg/L arsenic (NHW 1978). Taking into account this limit and the arsenic intake from other dietary sources, it can be seen that drinking water could become a major source of arsenic intake.

There is evidence which indicates that arsenic is a carcinogenic substance to humans. Analysis of the data is not sufficiently advanced to warrant lowering the level of arsenic in raw drinking water supplies, but this may occur in the future.

Although certain treatments remove arsenic from water (Cohen 1977; Carriker *et al.* 1976; Foley and Missingham 1977), at low concentrations these treatments are not very effective. Considering this and the drinking water standard, an objective of 0.05 mg/L of total arsenic is recommended for raw water supplies.

In the United States, the Environmental Protection Agency has recommended a limit of 0.05 mg/L arsenic in raw drinking water supplies (EPA 1976, p. 25). The WHO, Australian, French and Russian water quality criteria have the same maximum limit of 0.05 mg/L (Cottet 1975; Hart 1974, p. 48)

Aquatic Life and Wildlife

Aquatic Life

Concentrations of arsenic in Canadian waters were discussed in the section Occurrence, Forms and Sources.

Arsenic can occur in a number of forms in the aquatic environment. It is thought that most groups of organisms can synthesize organic arsenic from the inorganic forms (Bryan 1976, p. 20). Inorganic arsenic (as As-arsenic acid) administered to the gut of brown trout (*Salmo trutta*) was converted to (nonanionic) organic arsenic and appeared within one day in the gastrointestinal tract of the fish. This form of arsenic seemed to be selectively absorbed and stored in the tissues when both forms were present. Both inorganic and organic arsenic were excreted in the bile (Penrose 1975).

Arsenic compounds are reduced and methylated by anaerobes to give the organo-arsenicals dimethylarsine and trimethylarsine as volatile products of extreme toxicity. These arsines are readily oxidized to give products which are less toxic, such as cacodylic acid, $AsO_2H(CH_3)_2$ (Wood 1974).

It has been stated in the literature that pentavalent arsenicals are generally less toxic than trivalent ones which specifically block lipoate dependent enzymes (Frost 1967). This does not appear to be valid for acute toxicity, as the Merck Index stated that the acute toxicity from an oral dose to rats was 14 mg/kg arsenite and 14 mg/kg arsenate (Sundaram, personal communication). The salt will also govern the acute toxicity, as the LD_{50} for sodium arsenite given orally to rats is 41 mg/kg and for potassium arsenite, 14 mg/kg (NIOSH 1974 p. 77). The relative toxicity of the two valency forms in chronic toxicity is uncertain (Sundaram, personal communication). Trivalent arsenicals appear to be oxidized in living tissue to pentavalent compounds. The difference in toxicity of arsenicals is partially related to the rate of elimination from the body (Frost 1967).

Arsenic is fairly persistent in water, and in one experiment, 20% to 40% was still present in water 16 weeks after application (Gilderhus 1966).

Arsenic has been shown to be more toxic to plants than to animals. Thus it is used in some herbicides for the control of aquatic plant growth. Two to 5.0 mg/L (1.5 to 3.8 mg/L as As) of arsenic trioxide, used to control vegetation in ponds, was considered safe for fish (Becker and Thatcher 1973). Kemp *et al.* (1966) stated that 1.0, 2.0 and 4.0 mg/L sodium arsenite did not inhibit algal growth completely.

Gilderhus (1966) studied the effects on organisms of different levels of sodium arsenite and different methods of dosing, using concrete pools containing sediments, plants, invertebrates and fish. In the pools that received doses ranging from 0.69 to 2.76 mg/L arsenic, fewer fish survived than in the control pools. There was 18% survival for immature fish in the pool receiving 11.07 mg/L arsenic (in doses of 0.69 mg/L once weekly) over the experimental period of 16 weeks compared with 90% survival in the control pool. Among the adult bluegills 31% of those receiving arsenic survived, whereas 60% of the adult fish survived in the control pool. Of the survivors from ponds dosed with arsenic, there was a lower weight gain in the immature fish than in the control fish and the adults in the higher concentration lost weight. There was no significant difference between the weight of adults exposed to 0.69 and 0.23 mg/L arsenic as $NaAsO_2$ applied only once, 0.23 mg/L applied once monthly (total 0.92 mg/L As) and 0.023 mg/L arsenic applied weekly over 16 weeks (total 0.37 mg/L As), and the controls. Reproduction was affected by the arsenic, as development of the ova was prevented by the higher concentrations. In this experiment the adult fish were more tolerant than the immature fish. Holland *et al.* (1960) also found that younger fish were less tolerant than older fish.

In another study the macroinvertebrates in pools were reduced in numbers when the treatment over the year totalled 4.0 mg/L or more of arsenic (Gilderhus 1966). There was also a reduction in species diversity. The bottom fauna were affected in proportion to the amount of arsenic in the sediments. Examination of the fish after 16 weeks

showed that the digestive tract contained the highest level of arsenic, 17.6 mg/kg; liver, 11.6 mg/kg; kidney, 5.9 mg/kg; and ovaries, 8.4 mg/kg. The muscle contained 1.3 mg/kg in fish from the pool receiving one dose of 4.0 mg/L arsenic. The levels of 1.0 to 1.3 mg/kg arsenic in the muscle were associated with a significantly slower growth rate (Gilderhus 1966).

The toxicity of sodium arsenite was studied for a number of fish species. The 96-h LC_{50} varied between the different species of fish from 25.8 mg/L for brook trout to 72 mg/L for bluegill (at a water hardness of 140 to 152 mg/L as $CaCO_3$). Exposure beyond 96 h gave two types of toxicity curves. Arsenic became more toxic with time for bluegills, fathead minnows and channel catfish, but the LC_{50} became constant with time, 18 mg/L, after exposure of 262 hours for brook trout and 32 mg/L after 336 hours for goldfish. Both concentrations were approximately 30% lower than the 96-h LC_{50} (Cardwell *et al.* 1976). Sodium arsenite was shown to be more toxic than sodium arsenate (Grindley 1946).

Arsenic trioxide affected the weight and fat content of rainbow trout. Arsenic affected osmoregulation by causing kidney damage, and water was retained in the fish body; thus fish exposed to 1.0 mg/L arsenic lost dry weight but the wet weight remained unchanged. When exposed to 6.0 mg/L arsenic the wet weight of fish also decreased. Arsenic also affected the synthesis of lipids necessary as energy reserves and the development of eggs (Speyer and Leduc 1975). Since the statistical significance of the figures was not tested (Leduc, personal communication), only the qualitative aspect of the study has been discussed.

The eyed stage of rainbow trout (*Salmo gairdneri*) eggs was less sensitive than both young and adult fish and could withstand 50 mg/L of either arsenite or arsenate; these concentrations were reported to be lethal to the swimming stages (Dabrowski 1976). Arsenic (0.1 mg/L) as sodium arsenate significantly impaired the conditioned avoidance response of goldfish but this was not evident at 0.05 mg/L (Weir and Hine 1970).

The survival time of the green sunfish (*Lepomis cyanellus*) when exposed to arsenic decreased with increasing temperature. The LT_{50} decreased from 678 h at 10°C to 124 h at 30°C when the fish were exposed to 60 mg/L arsenic as sodium arsenate. The LT_{50} at 30°C for 30 mg/L arsenic was 209 h (Sorensen 1976a).

An increase in temperature increased the susceptibility of the rotifer *Philodina roseola* to sodium arsenate. The 48-h TL_m was 78 mg/L at 5°C, 57 mg/L at 15°C, and 44 mg/L at 20°C. Temperature had less effect on the susceptibility of *Philodina* over a longer exposure time. The 72-h TL_m was 29 mg/L at 5°C, 26 mg/L at 15°C, and 21 mg/L at 20°C (Schaefer and Pipes 1973).

Birge *et al.* (1974) found indications that homeotherms were more susceptible to arsenic, as sodium arsenite, than were poikilotherms. There was a 65% hatch in chick embryos at 0.001 mg/L arsenic (administered by yolk sac injection to give the specified concentration) and a 96% and 47% hatch at 0.1 and 0.75 mg/L, respectively, with trout embryos (*Salmo gairdneri*). Gross abnormalities occurred in 27% of the trout embryos surviving exposure to 1.0 mg/L arsenic, in 11% surviving exposure to 0.25 mg/L, and in 2% surviving exposure to 0.025 mg/L, indicating that arsenic has teratogenic properties. Arsenic oxides have been shown to affect oxidative phosphorylation and as there is a higher rate of oxidative metabolism in homeotherms, this could be one reason for the higher sensitivity of the chick embryos.

A 16% reduction in *Daphnia magna* reproduction occurred at 0.52 mg/L arsenic as Na_2HAsO_4 over a period of 3 weeks. The 3-week LC_{50} was 2.85 mg/L and the 48-h LC_{50} was 7.4 mg/L. The percentage of protein as an estimation of growth was less in *Daphnia* exposed to arsenic compared with the controls (Biesinger and Christensen 1972). Macroinvertebrates varied in susceptibility to arsenic; midges were less tolerant than May flies and dragonflies (Rudolfs *et al.* 1950).

Using waters of varying hardness (13.0, 52.2, 208.7 and 305.2 mg/L as $CaCO_3$), Inglis and Davis (1972) determined that hardness did not have a significant effect on toxicity. The 96-h LC_{50} for arsenic as $NaAsO_2$ for bluegills (*Lepomis macrochirus*) was 26.5 mg/L in soft water, 28.0 mg/L in medium hard water and 26.7 mg/L in hard water.

There are a number of studies giving the levels of arsenic which have been measured in aquatic organisms. Arsenic is accumulated from water by organisms, but there does not seem to be biomagnification through the food web. Seydel (1972) gave levels of 0.0016 mg/L in water, 15.2 mg/kg in sediments, 6.7 mg/kg in benthos, 5.8 mg/kg in phytoplankton and 6.2 mg/kg in zooplankton. Lunde (1970) also found no indication of biomagnification. Predators on zooplankton, benthos and fish contained levels of 3.4, 5.0 and 2.2 mg/kg arsenic, respectively.

No evidence of biomagnification through the food web was found by Isensee *et al.* (1973). Algae and *Daphnia* bioaccumulated more cacodylic acid and dimethylarsine than snails and fish.

Levels of arsenic in dressed fish from Lakes Erie and Ontario and Moose Lake ranged from <0.05 to 0.09 mg/kg wet weight (Uthe and Bligh 1971). Falk *et al.* (1973)

reported arsenic concentrations in the liver of pike and walleye from Grace Lake, NWT, as 0.96 and 1.62 mg/kg, respectively, and in the muscle as 1.69 and 0.29 mg/kg, respectively. Concentrations in fish from areas which did not have gold mines were lower. The biological half-life in the liver and gastrointestinal tract was approximately one week (Sorensen 1976a). The average for arsenic in edible parts of freshwater fish and fishery products throughout Canada varied from 0.01 to 0.62 mg/kg. Fish most likely to accumulate arsenic are those living near or at the bottom (CPHA 1977).

The bioaccumulation ratios (concentration in tissue to concentration in water) for algae, crayfish, *Daphnia*, mosquito fish and catfish were 34, 5, 4, 130 and <1, respectively. Aquatic macrophytes ranged from 110 to 14 500, the emergent parts containing less arsenic than the submerged parts. Levels of arsenic in algae ranged from 3 to 7000 times the concentration in water. Marine plants had higher concentrations than freshwater plants (Woolson 1975). Different species of aquatic macrophytes growing in the same location accumulated different amounts of arsenic from water containing <0.1 mg/L arsenic. *Potamogeton cheesemanii* contained 45 mg/kg (air-dried); *Ceratophyllum demersum*, 165 mg/kg; and *Egeria densa*, 310 mg/kg. Plants from different areas of the river, which contained up to 1.0 mg/L arsenic from geothermal sources, had levels as high as 1450 mg/kg (*Lagarosiphon major*) (Lancaster *et al.* 1971). Arsenic was present in the algae and fish as water-soluble and lipid-soluble arseno-organic compounds. Arsenic fed in contaminated food did not appear to be toxic, as most of it was excreted (Woolson 1975).

The concentration of arsenic in fish was positively correlated to the concentration of arsenic in the water. No correlation was found between the accumulated arsenic and the fish length, weight or condition (Sorensen 1976b). Bohn and McElroy (1976) found a positive correlation between levels of arsenic in whole fish (marine Arctic cod) and the body fresh weight. The muscle contained a mean of 46 mg/kg arsenic (dry weight) and the liver, 4.4 mg/kg (dry weight). This distribution contrasted with that found in freshwater fish by Gilderhus (1966) where the muscle contained less than the liver. Copepods, a major component of Arctic cod diet, contained less arsenic (5.6 to 7.3 mg/kg) than the predator's muscle. This does not necessarily indicate biomagnification, as the diet of the cod changed with age (Bohn and McElroy 1976).

Very little information was found on antagonistic or synergistic effects of arsenic with other elements. Arsenic decreased the toxic effects of selenium and caused an increase in selenium excretion in the bile but the mechanism was unknown (NAS 1976, p. 65). Frost (1967) stated that arsenicals are capable of counteracting the toxicity of selenium, but that the action depends on the level of each element.

Recommended Objective for Aquatic Life

There is very little information on the sublethal effects of arsenic on aquatic organisms. Since a unimodal form of toxicity was not found in some of the fish tested and the toxicity increased with time, extrapolation from the available acute toxicity studies to give a safe level has not been attempted.

Arsenic has been shown to be persistent in water and to be bioaccumulated by aquatic plants and animals, but there is no evidence of biomagnification through the food web. Arsenic fed as contaminated food did not appear to be toxic, since most of it was eliminated from the body.

The level of arsenic recommended for public water supplies (0.05 mg/L) is below levels of arsenic that have been shown to have no detrimental effects on aquatic organisms and thus should provide a safe level for aquatic organisms.

Wildlife

No information was found in the literature which could be used to recommend a guideline for wildlife. It may prove possible to use the level of 1.0 mg/L total arsenic recommended for livestock water supplies (see next section), but at present there is not enough information.

Livestock Water Supplies

Toxicity

Wadsworth (1952) has listed the following arsenic toxic doses for farm animals: poultry, 0.05 to 0.1 g per bird; dogs, 0.1 to 0.2 g per animal; pigs, 0.5 to 0.1 g per animal; sheep, goats and horses, 10 to 15 g per animal; and cattle, 15 to 30 g per animal. The lethal dose for animals is believed to be approximately 44 mg/kg of body weight (Luh *et al.* 1973).

Some arsenic compounds have been shown to have therapeutic and growth-promoting properties and have been approved for use as feed additives for poultry and swine. The amounts permitted by the U.S. Food and Drug Administration range from 50 mg 3-nitro-4-hydroxyphenylarsonic acid (containing 14 mg arsenic) per kilogram of poultry feed to 375 mg of p-ureidobenzenearsonic acid (containing 108 mg arsenic) per kilogram of turkey feed. The maximum levels of arsenic in tissues ranged from 0.5 mg As/kg of fresh muscle to 2.0 mg As/kg of fresh uncooked kidney and liver (Calvert 1975). Data quoted by Calvert (1975) showed that the accumulation of arsenic in various organs was proportional to the amount ingested. Thus chicks fed a diet containing 34 mg As/kg (as arsa-

nilic acid) for nine weeks had a fresh liver level of 0.9 mg As/kg, while chicks fed a diet containing ten times as much arsenic had a fresh liver level of 4.8 mg As/kg. Similarly, chicks fed a diet containing 14 mg As/kg (as 3-nitro-4-hydroxyphenylarsonic acid) had a liver level of 1.8 mg As/kg, while chicks fed a diet containing 140 mg As/kg of this compound had a liver level of 5.7 mg As/kg. Comparing this set of numbers with the previous values, it is evident that the accumulation of arsenic depends also on its chemical form.

The ability of chick embryos to hatch was seriously affected, the survival rate being 65% of controls, at an As^{3+} concentration in the egg yolk of 0.001 mg/kg. At higher concentrations there was a teratogenic effect and defective survivors appeared (Birge and Roberts 1976). The accumulation of arsenic by animal tissues is dependent on the animal type. Rats on a diet containing 39 mg As/kg had a blood concentration of 125 mg/L after 21 days. Concentrations in other organs or tissues were: spleen, 60 mg/kg; heart, 43 mg/kg; skin, 27 mg/kg; kidney, 25 mg/kg; liver, 20 mg/kg (Peoples 1964). Another study with rats fed a diet containing arsenic resulted in even higher organ and tissue concentrations, although neither growth rate nor survival and longevity were affected and no pathological changes were observed (Schroeder *et al.* 1968). A diet containing 39 mg/kg arsenic fed to hamsters, guinea pigs, and rabbits gave completely different results. The highest organ (tissue) concentrations in these cases were: skin, 38 mg/kg in hamster; heart, 20 mg/kg in guinea pig; and skin, 2.5 mg/kg in rabbit (Peoples 1964). Rabbits with an arsenic intake of 2.5 mg/kg showed disturbance in their muscular coordination, morphological changes in the red blood cells, and kidney impairment (Akulou *et al.* 1959).

In a study by Peoples (1964) it was found that very little or no arsenic fed as arsenic acid was stored in the tissue of cows or in their milk. Most of the intake of arsenic was eliminated rapidly in the urine. The remaining amount depleted rapidly when the feeding of arsenic was stopped. Dickinson (1972) reported that clinical signs of arsenic poisoning, diarrhea and anorexia appeared in cattle given a total dose of 50 to 70 mg monosodium acid methanearsonate (MSMA), containing 25 to 35 mg arsenic per kilogram of body weight. The MSMA was administered orally in doses of 10 mg/kg of body weight per day. A total of 100 mg MSMA per kilogram of body weight was fatal to four of the five cows used in the experiment. Kidney failure or toxic nephrosis was the apparent cause of death.

Studies with sheep on a diet containing arsenic showed that the liver and kidney had the highest arsenic concentration. Thus, a diet containing 26.8 mg As/kg led to liver and kidney arsenic concentrations of 3.1 and 3.2 mg/kg, respectively (muscle concentration, 0.2 mg/kg; blood, 0.063 mg/L). As in the case of chicks discussed earlier, the concentration of arsenic in the tissues was proportional to the concentration of arsenic in the feed. A feed concentration of 144.4 mg/kg gave liver and kidney levels of 26.8 and 12.2 mg/kg, respectively. The elimination of arsenic was quite rapid when the administration of feed containing arsenic was stopped. The liver arsenic level of 26.8 mg/kg mentioned above dropped to 15.4 mg/kg after 2 days and to 3.5 mg/kg after 6 days. Five-day balance trials showed that 76% to 91% of the arsenic contained in feed was excreted (Calvert 1975).

Recommended Objective for Water Supply for Livestock

The available evidence shows that arsenic does not accumulate to any great extent in animal tissues and it is well tolerated at diet levels of 10 to 20 mg/kg (Frost 1967). The U.S. National Academy of Sciences (NAS 1973, p. 310) has recommended an upper limit of 0.2 mg/L in the water supply for livestock enterprises. The Australian Water Quality Criteria (Hart 1974, p. 122) recommend a limit of 1 mg/L.

Since the surface waters in Canada very seldom had a dissolved arsenic concentration greater than 0.05 mg/L, an objective of 0.5 mg/L as total arsenic in water is recommended for farm animals.

Irrigation Water

Arsenic is widespread in natural soils. The arsenic concentration in 207 soil samples from the agricultural areas in Ontario, where no arsenic compounds had been used, ranged from 1.1 to 16.7 mg/kg, with an average of 6.27 mg/kg. In 31 samples of apple orchard soils, where pesticides containing arsenic have been used for periods of up to 70 years, the mean arsenic level in the top 15 cm of soil was 54.2 mg/kg, with a range of 3.2 to 126 mg/kg. At the 15 to 30 cm level the range was 3.9 to 55.8 mg/kg, with an average of 20.9 mg/kg. It was also observed that the concentration of arsenic in both surface and subsurface soils increased exponentially with the age of the orchard (or with the period of time arsenic pesticides had been applied to the soil) (Frank *et al.* 1976a, 1976b). The average arsenic concentration in a variety of soil samples collected in southwestern Ontario was between 5.1 and 6.3 mg/kg, with a range of 1.2 to 20.0 mg/kg (Whitby *et al.* 1978). Values of 0.2 to 40 mg/kg have been quoted for arsenic concentrations in uncontaminated U.S. soils, and up to 550 mg/kg for arsenic-treated soils (Walsh and Keeney 1975). Frank *et al.* (1976b) found that arsenic accumulated in soils where arsenic compounds (e.g., lead arsenate, sodium arsenite) had been used as pesticides. The amount accumulated was a function of the length of time the arsenic compounds had been used on the respective soil and the amount sprayed. The amount of arsenic accumulated varied also with the type of soil (Woolson

1973) and the depth (Johnson and Hiltbold 1969; Frank *et al.* 1976b). The concentration decreased with depth and most of the arsenic accumulated in the top 30 cm of soil. In the case of known arsenic sources, such as a smelter operation, it was found that the concentration of arsenic in soil was the highest near the source and that it decreased with increasing distance from it (Amasa 1975). An approximate calculation using the number given by Deuel and Swoboda (1972) showed that only about 1% or less of the applied arsenic could be recovered as the "extractable" or "water soluble" form. Porter and Peterson (1975) gave a ratio of approximately 1:10 000 for water soluble arsenic: total soil arsenic. The rest was presumably tied down by soil particles and soil compounds. The adsorption of a series of organoarsenicals increased with the soil clay content (Hiltbold 1975). The initial symptom of arsenic toxicity to plants is wilting of new-cycle leaves, which is followed by retardation of root and top growth of the plant (Liebig 1966). Arsenic accumulates in or on the roots, the tips of new, fine roots being affected first. Apparently the effect of arsenic toxicity is such that the plant growth is limited before large amounts of arsenic are absorbed and translocated to the top. Toxic amounts of arsenic arrest the germination of seeds. The arsenic toxicity has a tendency to persist for several years, even when no more arsenic is added. Liebig (1966) has classified a series of vegetables and small fruits according to their tolerance to water-soluble arsenic into three groups: (i) very tolerant (asparagus, potato, tomato, carrot, tobacco, strawberry, grape, red raspberry); (ii) fairly tolerant (strawberry and sweet corn on heavy and medium soils, beet, squash), and (iii) low or no tolerance (snap bean, lima bean, onion, pea, cucumber, alfalfa, sweet corn and strawberry on light, sandy soils). Some of the tolerant crops—potatoes, corn, rye, wheat, lemon plants—exhibit small yield increases at low levels of arsenic.

Toxicity of arsenic, expressed as loss of growth, correlated slightly better with the various types of "soluble" or "extractable" arsenic than with the total arsenic (Woolson *et al.* 1971; Deuel and Swoboda 1972). Critical levels of arsenic ranged from 25 to 85 mg/kg for total arsenic and from 3 to 28 mg/kg for water-soluble arsenic. The levels were dependent on the soil type, the plant species (Walsh and Keeney 1975), and the arsenic compounds used (Woolson *et al.* 1971). For example, of a series of arsenates studied, NaH_2AsO_4 was the most toxic and $Fe(H_2AsO_4)_3$, the least toxic. The latter had only 13% of the toxicity of the former as measured by the GR_{50} values for corn (GR_{50} for NaH_2AsO_4 was 42 mg/kg and GR_{50} for $Fe(H_2AsO_4)_3$ was 325 mg/kg). The GR_{50} values of corn for calcium and aluminum arsenates had intermediate values (Woolson *et al.* 1971). The GR_{50} is the soil arsenic concentration at which the growth is reduced to 50% of that obtained on a soil not treated with an arsenic compound.

Very little or no arsenic was found in the edible parts of plants such as snap beans, sweet corn, peas or potatoes. In this case the danger of arsenic rests in decreased soil productivity rather than in injury to the consumer from any food crops produced (Jacobs *et al.* 1970). Potato peelings, however, had quite high arsenic concentrations, ranging from 2.9 mg/kg at a total arsenic concentration in soil of 11 to 19 mg/kg (corresponding extractable arsenic was 2.7 to 4.9 mg/kg) to 83 mg/kg at a total arsenic concentration in soil of 150 to 250 mg/kg (corresponding extractable arsenic was 53 to 68 mg/kg). "Extractable" arsenic was measured by extracting the sample with Bray P-1 solution (0.025 *N* HCl + 0.3 *N* NH_4F).

In a study (Woolson 1973) of six vegetable crops (green bean, lima bean, spinach, cabbage, tomato and radish), the GR_{50} of available arsenic varied from 6.2 mg/kg for green beans to 48.3 mg/kg for cabbage. "Available" arsenic in this case was measured by extracting the soil with 0.05 *N* H_2SO_4 and 0.025 *N* HCl. Four of the crops—green beans, lima beans, cabbage, tomatoes—showed very little tendency to accumulate arsenic. The arsenic content of the edible dry-plant parts at GR_{50} ranged from 0.7 mg/kg for tomatoes (GR_{50} = 25.4 mg/kg) to 4.2 mg/kg for green beans. The arsenic concentration in the dry edible part of spinach was 10 mg/kg, about the same as the soil concentration (10.6 mg/kg available arsenic) in which it was grown. Only radish had a high arsenic concentration, 76 mg/kg (dry basis), compared with the available arsenic in the soil, which was only 19.0 mg/kg (on a wet weight basis, the radish concentration was 8 mg/kg). This was explained by the fact that radish is a root crop and the radishes were not peeled (Woolson 1973). Arsenic was also toxic to cotton and soybean plants. Water-soluble arsenic levels detrimental to soybeans were 3 and 12 mg/kg on two different types of soil. The water-soluble arsenic levels detrimental to cotton were 8 and 28 mg/kg soil, again on two different types of soil. The lower concentrations corresponded to a fine, sandy loam (Amarillo type) and the higher value, to Houston Black clay (Deuel and Swoboda 1972).

Recommended Objective for Irrigation Water

It seems that soil concentrations of 5 mg/kg total arsenic are toxic to oilseed crops on fine sandy loam soil, and concentrations of 25 mg/kg or higher are toxic to less sensitive crops. On clay soil the corresponding values are approximately 20 and 50 mg/kg.

Assuming that approximately 1 m^3 of water is used for irrigating 1 m^2 per year (NAS 1973, p. 340) and that the

arsenic in the water will accumulate in the soil over a ten-year period, then the recommended objectives for irrigation waters are as follows (see Appendix A).

Type of crop	Type of soil	
	Sandy loam	Clay
Sensitive to arsenic	0.1 mg/L	1.0 mg/L
Tolerant to arsenic	1.0 mg/L	2.0 mg/L

These values incorporate a series of safety factors, as (i) only soils with high clay content will adsorb all or almost all the arsenic present in the water (Hiltbold 1975), (ii) it has been considered that arsenic accumulates only in the top 15 cm of soil. In fact part of the arsenic will also accumulate at depths greater than 15 cm, (iii) only a fraction of the total arsenic present in the soil is available to the plants, and (iv) over a long period of time some of the arsenic will be removed from the soils by the crops themselves or by physico-chemical processes. On the other hand, these recommended objectives do not take into consideration (i) the natural levels of arsenic present in soil and (ii) other arsenic sources. If in given areas natural arsenic levels are high, e.g., above 5 mg/kg, the recommended objectives for irrigation waters will have to be lowered. The recommended objectives will also have to be lowered if in addition to arsenic from irrigation waters there are other arsenic inputs to the area under consideration, such as usage of arsenical pesticides or industrial activities with arsenic emissions.

In the United States the EPA has recommended limits of 0.10 mg/L arsenic for continuous use on all soils and 2 mg/L for use up to 20 years on fine-textured neutral to alkaline soils (NAS 1973, p. 341). The Australian Water Quality Criteria recommend a limit of 0.1 mg/L (Hart 1974).

Recreation and Aesthetics

Potable water supplies in recreational areas should comply with the level of drinking water of 0.05 mg/L arsenic. Since arsenic has not been shown to biomagnify, the fish in these areas would probably not contain levels of arsenic which could be a hazard to public health. Also it was found that arsenic consumed from contaminated food was eliminated fairly rapidly from the body.

Recommended Objective for Recreational Waters

The recommended objective for recreational waters is 0.05 mg/L arsenic.

Industrial Water Supplies

The only industry in which the concentration of arsenic in the process water is of importance is the food industry.

The general principle that the water which is incorporated in the final product should meet the same requirements as for drinking water applies here also.

Recommended Objective for Industrial Water Supplies

A maximum of 0.05 mg/L arsenic is recommended for water which is incorporated in the final product in the food industry. This includes water used for the cooling of cans in the canning industry.

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Appendix A

Calculation of Recommended Limits for Arsenic Concentration in Irrigation Water

Assumptions

1. *Toxic levels*

Type of crop	Type of soil	
	Sandy loam	Clay
Sensitive to arsenic	5 mg/kg	20 mg/kg
Tolerant to arsenic	25 mg/kg	50 mg/kg

2. Amount of irrigation water used: 1000 L/m^2 per year

3. All arsenic in the water will accumulate in the top 15 cm of soil over a ten-year period.

4. Density of soil: 1.5 g/cm^3

Calculations

1. Weight of 1 m^2 of soil, 30 cm deep:

   $1.5\ g/cm^3 \times 10^4\ cm^2 \times 15\ cm = 225\ kg$

2. Amount of irrigation water used on 1 m^2 over a ten-year period: 10^4 L

3. Recommended limit for sensitive crop on sandy loam soil is then given by:

   $5\ mg/kg \times 225\ kg/10^4\ L \simeq 0.1\ mg/L$

   The calculations for other types of crops and soils are similar. The only value which changes is the toxic level.